abrupt down-slope of the voluntarily fatigued muscle is due to the greater consumption of its "reserve elements," apart from the production of lactic acid (I. Rauke). He asks the question, accordingly, whether the relaxation of a muscle in general takes place more slowly when its reserve elements are fewer. This he investigates by an experiment, the conditions of which are to compare the curve of a voluntarily fatigued muscle with that of a muscle whose excitability is reduced by lactic-acid artificially, i. e., in such a way as not to reduce the "reserve-elements" of the preparation. He injected the gastrocnemius of a frog with a .125% lactic acid solution. Its down-slope, like that of the cooled muscle, was less steep than that of the normally fatigued muscle. The same resulted with muscles injected with .1 to .2% soda. The rest of the paper is devoted to a discussion of theories of the molecular processes of muscular contraction and relaxation.

SCHENCK, *Ein apparat zur Verzeichnung von Länge und Spannung des Muskels*, Ibid, p. 108.

The title of this article indicates its contents.

WALLER, *On the Inhibition of Voluntary and Electrically Excited Muscular Contraction by Peripheral Excitation*, *Brain*, LVII., 1892, 35.

Waller asks whether the diminution in the force of a voluntary muscular contraction brought about by the superposition of direct electrical excitation is due to central inhibition (Fick), or to peripheral inhibition in the body of the muscle (Mosso), or to the excitation of the antagonists. Experimenting on the flexor muscles of the forearm and taking records, both by the dynamograph and "bag recorder" (see description in Brain, 1891, 206), he finds that there is a diminution in the maximum voluntary longitudinal effect, due to the electrical stimulation, but an increase in the maximum voluntary lateral effect. Further, that voluntary contraction superposed upon maximum direct foridization increases both the longitudinal and lateral effects, but that while the sense of maximum direct plus voluntary longitudinal effects is less than the maximum voluntary alone, the sense of the maximum direct plus voluntary lateral effects is more than the maximum of either taken alone. He explains these results largely by the stimulation of the antagonists (extensors); supporting this view by researches on the elevator muscles of the lower jaw, which have no antagonists (in which case the phenomenon in question does not appear), and on the flexors and extensors of the arm (which when both directly stimulated reproduce the phenomenon). A farther question is: Does the cessation of voluntary contraction involve simply a cessation of central voluntary emission, or a stimulation of the antagonists? Waller holds that the former is the main effect. He finds that in cases where the antagonist (extensor) is contracted, there is, on the release of the muscle (flexor), a prolongation of the lateral effect as compared with the longitudinal effect; but in voluntary release of the flexors, there is no such prolongation of the lateral effect. He also finds that in simultaneously grasping with one hand and letting go with the other, the two effects (curves) begin to appear simultaneously; but if the agent in each process be foridization, the muscle stimulated begins to contract before the released muscle begins to cease contracting. Turning from the effects of induced to those of galvanic currents in connection with voluntary contraction, he reaches conclusions which confirm Pflüger's and Waller and De Walleville's earlier results (Phil. Trans., Royal Soc., 1882). The general result is that "active arrest of action, i. e., true physiological inhibition of voluntary muscle, has not yet been demonstrated."